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| NEWS EXPRESS | OCTOBER 29 CURRENT WINDOWS VERSION IS V7.01A, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 11 AUGUST 2004 |
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FILE 'HOME' ENTERED AT 13:55:14 ON 26 NOV 2004

=> FIL MEDLINE BIOSIS EMBASE CA SCISEARCH

| COST IN U.S. DOLLARS | SINCE FILE ENTRY | TOTAL SESSION |
|---|---|---|
| FULL ESTIMATED COST | 0.21 | 0.21 |

FILE 'MEDLINE' ENTERED AT 13:55:24 ON 26 NOV 2004

FILE 'BIOSIS' ENTERED AT 13:55:24 ON 26 NOV 2004


FILE 'EMBASE' ENTERED AT 13:55:24 ON 26 NOV 2004

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FILE 'CA' ENTERED AT 13:55:24 ON 26 NOV 2004
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FILE 'SCISEARCH' ENTERED AT 13:55:24 ON 26 NOV 2004
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=> s ((phosphatidylinositol or PI) (n) 3 (n) kinase? (n) (p110 or delta)) or (PI3
kinase? (n) (p110 or delta)) or (PI3K? (n) (p110 or delta))
L1          571 ((PHOSPHATIDYLINOSITOL OR PI) (N) 3 (N) KINASE? (N) (P110 OR
                DELTA)) OR (PI3 KINASE? (N) (P110 OR DELTA)) OR (PI3K? (N) (P110
                 OR DELTA))

=> s antisense or (anti (n) sense) or (complemen? (2n) (oligonucl? or nucle?))
L2       145712 ANTISENSE OR (ANTI (N) SENSE) OR (COMPLEMEN? (2N) (OLIGONUCL?
                OR NUCLE?))

=> s l1 and l2
L3           22 L1 AND L2

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4            9 DUP REM L3 (13 DUPLICATES REMOVED)

=> s MONIA, B?/au; s COWSER, L?/au
L5          906 MONIA, B?/AU


S COWSER, IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s MONIA, B?/au;s COWSER, L?/au
L6          906 MONIA, B?/AU


S COWSER, IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s MONIA, B?/au;s COWSERT, L?/au
L7          906 MONIA, B?/AU


S COWSERT, IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s cowsert, L?/au
L8          438 COWSERT, L?/AU

=> s l5 or l8
L9         1233 L5 OR L8

=> d his'
'HIS'' IS NOT A VALID FORMAT
In a multifile environment, a format can only be used if it is valid
in at least one of the files.  Refer to file specific help messages
```

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or the STNGUIDE file for information on formats available in
individual files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):end

=> end
ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
LOGOFF? (Y)/N/HOLD:quit
'QUIT' IS NOT VALID HERE
For an explanation, enter "HELP LOGOFF".

=> d his

     (FILE 'HOME' ENTERED AT 13:55:14 ON 26 NOV 2004)

     FILE 'MEDLINE, BIOSIS, EMBASE, CA, SCISEARCH' ENTERED AT 13:55:24 ON 26
     NOV 2004
L1          571 S ((PHOSPHATIDYLINOSITOL OR PI) (N) 3 (N) KINASE? (N) (P110 OR
L2       145712 S ANTISENSE OR (ANTI (N) SENSE) OR (COMPLEMEN? (2N) (OLIGONUCL?
L3           22 S L1 AND L2
L4            9 DUP REM L3 (13 DUPLICATES REMOVED)
L5          906 S MONIA, B?/AU
L6          906 S MONIA, B?/AU
L7          906 S MONIA, B?/AU
L8          438 S COWSERT, L?/AU
L9         1233 S L5 OR L8

=> s l9 and l1
L10           4 L9 AND L1

=> s l4 or l10
L11          11 L4 OR L10

=> dup rem l11
PROCESSING COMPLETED FOR L11
L12           9 DUP REM L11 (2 DUPLICATES REMOVED)

=> d l12 ibib abs

L12  ANSWER 1 OF 9     MEDLINE on STN
ACCESSION NUMBER:   2002408450     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 12162696
TITLE:              GeneBlocs are powerful tools to study and delineate signal
                    transduction processes that regulate cell growth and
                    transformation.
AUTHOR:             Sternberger Maria; Schmiedeknecht Anett; Kretschmer Anny;
                    Gebhardt Frank; Leenders Frauke; Czauderna Frank; Von
                    Carlowitz Ira; Engle Mike; Giese Klaus; Beigelman Leonid;
                    Klippel Anke
CORPORATE SOURCE:   atugen AG, Berlin, Germany.
SOURCE:             Antisense & nucleic acid drug development, (2002 Jun) 12
                    (3) 131-43.
                    Journal code: 9606142. ISSN: 1087-2906.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        200301
ENTRY DATE:         Entered STN: 20020807
                    Last Updated on STN: 20030123
                    Entered Medline: 20030122
AB   The study of signal transduction processes using antisense
     oligonucleotides is often complicated by low intracellular stability of
     the antisense reagents or by nonspecific effects that cause
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toxicity. Here, we introduce a new class of **antisense** molecules, so-called GeneBlocs, which are characterized by improved stability, high target RNA specificity, and low toxicity. GeneBlocs allow for efficient downregulation of mRNA expression at nanomolar concentrations, and they do not interfere with cell proliferation. We demonstrate these beneficial properties using a positive readout system. GeneBloc-mediated inhibition of tumor suppressor PTEN (phosphatase and tension homologue detected on chromosome 10) expression leads to hyperactivation of the phosphatidylinositol (PI) 3-kinase pathway, thereby mimicking the loss of PTEN function and its early consequences observed in mammalian cancer cells. Specifically, cells treated with PTEN GeneBlocs show functional activation of Akt, a downstream effector of PI 3-kinase signaling, and exhibit enhanced proliferation when seeded on a basement membrane matrix. In addition, GeneBlocs targeting the catalytic subunit of **PI 3-kinase**, **p110**, specifically inhibit signal transduction of endogenous or recombinant PI 3-kinase. This demonstrates that GeneBlocs are powerful tools to analyze and to modulate signal transduction processes and, therefore, represent alternative reagents for the validation of gene function.

=> d l12 ibib abs 2-9

L12 ANSWER 2 OF 9 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
DUPLICATE 1
ACCESSION NUMBER: 2001:253065 BIOSIS
DOCUMENT NUMBER: PREV200100253065
TITLE: **Antisense** modulation of **PI3 kinase p110** beta expression.
AUTHOR(S): **Monia, Brett P.** [Inventor]; **Cowsert, Lex M.** [Inventor]
CORPORATE SOURCE: ASSIGNEE: Isis Pharmaceutical Inc.
PATENT INFORMATION: US 6133032 October 17, 2000
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Oct. 17, 2000) Vol. 1239, No. 3. e-file. CODEN: OGUPE7. ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 23 May 2001
Last Updated on STN: 19 Feb 2002

AB **Antisense** compounds, compositions and methods are provided for modulating the expression of **PI3 kinase p110** beta. The compositions comprise **antisense** compounds, particularly **antisense** oligonucleotides, targeted to nucleic acids encoding **PI3 kinase p110** beta. Methods of using these compounds for modulation of **PI3 kinase p110** beta expression and for treatment of diseases associated with expression of **PI3 kinase p110** beta are provided.

L12 ANSWER 3 OF 9 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
DUPLICATE 2
ACCESSION NUMBER: 2000:447933 BIOSIS
DOCUMENT NUMBER: PREV200000447933
TITLE: **Antisense** modulation of **PI3 kinase p110** delta expression.
AUTHOR(S): **Monia, Brett P.** [Inventor]; **Cowsert, Lex M.** [Inventor]
CORPORATE SOURCE: ASSIGNEE: Isis Pharmaceuticals Inc.
PATENT INFORMATION: US 6046049 April 04, 2000
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Apr. 4, 2000) Vol. 1233, No. 1. e-file. CODEN: OGUPE7. ISSN: 0098-1133.

DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 18 Oct 2000
Last Updated on STN: 10 Jan 2002

AB **Antisense** compounds, compositions and methods are provided for modulating the expression of **PI3 kinase p110** delta. The compositions comprise **antisense** compounds, particularly **antisense** oligonucleotides, targeted to nucleic acids encoding **PI3 kinase p110** delta. Methods of using these compounds for modulation of **PI3 kinase p110** delta expression and for treatment of diseases associated with expression of **PI3 kinase p110** delta are provided.

L12 ANSWER 4 OF 9 MEDLINE on STN
ACCESSION NUMBER: 2000482758 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10850853
TITLE: IRAK-2 and PI 3-kinase synergistically activate NF-kappaB and AP-1.
AUTHOR: Guo F; Wu S
CORPORATE SOURCE: Institute of Pharmaceutic Sciences, The First Military Medical University, Guangzhou, China.
SOURCE: Inflammation, (2000 Aug) 24 (4) 305-16.
Journal code: 7600105. ISSN: 0360-3997.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200010
ENTRY DATE: Entered STN: 20001019
Last Updated on STN: 20001019
Entered Medline: 20001012

AB **Antisense** interleukin-1 (IL-1) receptor associated kinase-2 (IRAK-2) oligonucleotide (ODN) and **antisense p110 PI 3-kinase** ODN blocked IRAK-2 and **p110 PI 3-kinase** expression, respectively. As a result, **antisense** IRAK-2 ODN or **antisense p110 PI 3-kinase** ODN inhibited IL-1-induced NF-kappaB and AP-1 activation in HepG2 cells. The inhibition of NF-kappaB activation by **antisense** IRAK-2 ODN or **antisense p110 PI 3-kinase** ODN and the inhibition of AP-1 activation by **antisense** IRAK-2 ODN were incomplete, whereas AP-1 activation could be inhibited by **antisense p110 PI 3-kinase** ODN completely. These results indicate that IRAK-2 is necessary but insufficient to activate NF-kappaB and AP-1 completely and that although PI 3-kinase is not sufficient for NF-kappaB full activation, it is sufficient to activate AP-1 completely. The effects of IRAK-2 or PI 3-kinase on NF-kappaB and AP-1 activation were confirmed by the results that overexpression of IRAK-2 failed to fully activate NF-kappaB and AP-1 and that overexpression of **p110 PI 3-kinase** is insufficient for NF-kappaB full activation but sufficient for AP-1 activation. Cotransfection experiments showed that the combination of **antisense** IRAK-2 ODN and **antisense p110 PI 3-kinase** ODN resulted in additive inhibition of NF-kappaB as well as AP-1 activation. On the other hand, coexpression of IRAK-2 with **p110 PI 3-kinase** led to a synergistic activation of NF-kappaB and AP-1. These data suggest that IRAK-2 and PI 3-kinase cooperate to activate NF-kappaB and AP-1.

L12 ANSWER 5 OF 9 CA COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 130:151352 CA

TITLE: Distinct roles for the p110α and hVPS34 phosphatidylinositol 3'-kinases in vesicular trafficking, regulation of the actin cytoskeleton, and mitogenesis

AUTHOR(S): Siddhanta, Uma; McIlroy, James; Shah, Amishi; Zhang, Yitao; Backer, Jonathan M.

CORPORATE SOURCE: Department of Molecular Pharmacology, Albert Einstein College of Medicine, Bronx, NY, 10461, USA

SOURCE: Journal of Cell Biology (1998), 143(6), 1647-1659
CODEN: JCLBA3; ISSN: 0021-9525

PUBLISHER: Rockefeller University Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The authors have examined the roles of the p85/p110α and hVPS34 phosphatidylinositol (PI) 3'-kinases in cellular signaling using inhibitory isoform-specific antibodies. They raised anti-hVPS34 and anti-p110α antibodies that specifically inhibit recombinant hVPS34 and p110α, resp., in vitro. They used the antibodies to study cellular processes that are sensitive to low-dose wortmannin. The antibodies had distinct effects on the actin cytoskeleton; microinjection of anti-p110α antibodies blocked insulin-stimulated ruffling, whereas anti-hVPS34 antibodies had no effect. The antibodies also had different effects on vesicular trafficking. Microinjection of inhibitory anti-hVPS34 antibodies, but not anti-p110α antibodies, blocked the transit of internalized PDGF receptors to a perinuclear compartment, and disrupted the localization of the early endosomal protein EEA1. Microinjection of anti-p110α antibodies, and to a lesser extent anti-hVPS34 antibodies, reduced the rate of transferrin recycling in CHO cells. Surprisingly, both antibodies inhibited insulin-stimulated DNA synthesis by 80%. Injection of cells with **antisense** oligonucleotides derived from the hVPS34 sequence also blocked insulin-stimulated DNA synthesis, whereas scrambled oligonucleotides had no effect. Interestingly, the requirement for p110α and hVPS34 occurred at different times during the G1-S transition. These r data suggest that different PI 3'-kinases play distinct regulatory roles in the cell, and document an unexpected role for hVPS34 during insulin-stimulated mitogenesis.

REFERENCE COUNT: 64 THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 6 OF 9 CA COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 128:58975 CA

TITLE: Human phosphatidylinositol 3-kinase and its cloning, characterization, and enhanced expression in melanomas

INVENTOR(S): Vanhasebroeck, Bart; Waterfield, Michael Derek

PATENT ASSIGNEE(S): Ludwig Institute for Cancer Research, Switz.; Vanhasebroeck, Bart; Waterfield, Michael Derek

SOURCE: PCT Int. Appl., 72 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 9746688 | A1 | 19971211 | WO 1997-GB1471 | 19970530 |
| W: AU, CA, CN, JP, KR, NZ, US | | | | |
| RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE | | | | |
| CA 2256483 | AA | 19971211 | CA 1997-2256483 | 19970530 |
| AU 9729705 | A1 | 19980105 | AU 1997-29705 | 19970530 |
| AU 719354 | B2 | 20000504 | | |
| EP 914448 | A1 | 19990512 | EP 1997-924137 | 19970530 |

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI

| | | | | |
|---|---|---|---|---|
| CN 1220701 | A | 19990623 | CN 1997-195151 | 19970530 |
| NZ 332634 | A | 20000929 | NZ 1997-332634 | 19970530 |
| JP 2000517165 | T2 | 20001226 | JP 1998-500305 | 19970530 |
| US 6482623 | B1 | 20021119 | US 1998-194640 | 19981201 |
| US 2003099627 | A1 | 20030529 | US 2002-162160 | 20020603 |

PRIORITY APPLN. INFO.:

| | | |
|---|---|---|
| GB 1996-11460 | A | 19960601 |
| WO 1997-GB1471 | W | 19970530 |
| US 1998-194640 | A3 | 19981201 |

AB The invention relates to a novel lipid kinase termed p110δ which is part of the phosphatidylinositol 3-kinase (PI3) Kinase family. Human **PI3 Kinase p110δ** interacts with p85, has a broad phosphoinositide specificity, and is sensitive to the same kinase inhibitors as **PI3 Kinase p110** α. However in contrast to previously identified PI3 Kinases which show a ubiquitous pattern of expression, p110δ is selectively expressed in leukocytes. Importantly, p110δ shows enhanced expression in most melanomas tested and therefore may play a crucial role in regulating the metastatic property exhibited by melanomas. Further, p110δ undergoes autophosphorylation in a Mn2+-dependent manner, which hinders the lipid kinase activity of the protein. The identification of agents that enhance or reduce p110δ activity may therefore prevent cancer metastasis.

L12 ANSWER 7 OF 9 CA COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 125:140241 CA
TITLE: Association of phosphatidylinositol 3 kinase to protein kinase C ζ during interleukin-2 stimulation
AUTHOR(S): Gomez, Javier; Martinez-A., Carlos; Garcia, Alphonse; Rebollo, Angelita
CORPORATE SOURCE: Centro Nacional Biotecnologia, Universidad Autonoma, Madrid, E-28049, Spain
SOURCE: European Journal of Immunology (1996), 26(8), 1781-1787
CODEN: EJIMAF; ISSN: 0014-2980
PUBLISHER: VCH
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Interleukin-2 induces a serine-phosphorylated phosphatidylinositol 3 kinase activity in the mouse T cell line TS1αβ. Moreover, protein kinase C (PKC) ζ directly or indirectly assocs. with the phosphatidylinositol 3 kinase and the association appears to be necessary for the serine-phosphorylated phosphatidylinositol 3 kinase activity, since release of ζPKC by competition of binding with peptides spanning the p110 sequence from amino acids 907 to 925 abolishes the serine-phosphorylated phosphatidylinositol 3 kinase activity. This kinase activity is also blocked when ζPKC expression is inhibited by **antisense** oligonucleotide. Inhibition of phosphatidylinositol 3 kinase activity by wortmannin does not abolish ζPKC association

L12 ANSWER 8 OF 9 MEDLINE on STN
ACCESSION NUMBER: 96009592 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7565716
TITLE: A phosphatidylinositol (PI) kinase gene family in Dictyostelium discoideum: biological roles of putative mammalian p110 and yeast Vps34p PI 3-kinase homologs during growth and development.
AUTHOR: Zhou K; Takegawa K; Emr S D; Firtel R A
CORPORATE SOURCE: Department of Biology, Howard Hughes Medical Institute, University of California, San Diego, La Jolla 92093-0634, USA.

CONTRACT NUMBER: CA60559 (NCI)
SOURCE: Molecular and cellular biology, (1995 Oct) 15 (10) 5645-56.
Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U23476; GENBANK-U23477; GENBANK-U23478; GENBANK-U23479; GENBANK-U23480
ENTRY MONTH: 199510
ENTRY DATE: Entered STN: 19951227
Last Updated on STN: 20021218
Entered Medline: 19951025

AB Three groups of phosphatidylinositol (PI) kinases convert PI into PI(3)phosphate, PI(4)phosphate, PI(4,5) bisphosphate, and PI(3,4,5)trisphosphate. These phosphoinositides have been shown to function in vesicle-mediated protein sorting, and they serve as second-messenger signaling molecules for regulating cell growth. To further elucidate the mechanism of regulation and function of phosphoinositides, we cloned genes encoding five putative PI kinases from Dictyostelium discoideum. Database analysis indicates that D. discoideum PIK1 (DdPIK1), -2, and -3 are most closely related to the mammalian **p110 PI 3-kinase**, DdPIK5 is closest to the yeast Vps34p PI 3-kinase, and DdPIK4 is most homologous to PI 4-kinases. Together with other known PI kinases, a superfamily of PI kinase genes has been defined, with all of the encoded proteins sharing a common highly conserved catalytic core domain. DdPIK1, -2, and -3 may have redundant functions because disruption of any single gene had no effect on D. discoideum growth or development. However, strains in which both of the two most highly related genes, DdPIK1 and DdPIK2, were disrupted showed both growth and developmental defects, while double knockouts of DdPIK1 and DdPIK3 and DdPIK2 and DdPIK3 appear to be lethal. The delta Ddpik1 delta Ddpik2 null cells were smaller than wild-type cells and grew slowly both in association with bacteria and in axenic medium when attached to petri plates but were unable to grow in suspension in axenic medium. When delta Ddpik1 delta Ddpik2 null cells were plated for multicellular development, they formed aggregates having multiple tips and produced abnormal fruiting bodies. **Antisense** expression of DdPIK5 (a putative homolog of the Saccharomyces cerevisiae VPS34) led to a defect in the growth of D. discoideum cells on bacterial lawns and abnormal development. DdPIK5 complemented the temperature-sensitive growth defect of a Schizosaccharomyces pombe delta Svps34 mutant strain, suggesting DdPIK5 encodes a functional homolog of yeast Vps34p. These observations indicate that in D. discoideum, different PI kinases regulate distinct cellular processes, including cell growth, development, and protein trafficking.

L12 ANSWER 9 OF 9 CA 
ACCESSION NUMBER: 120:100544 CA
TITLE: Cloning and expression of a cDNA for a subunit of PI3 kinase
INVENTOR(S): Hiles, Ian D.; Fry, Michael J.; Dhand, Ritu; Waterfield, Michael D.; Parker, Peter J.; Otsu, Masayuki; Panayotou, George; Volinia, Stefano; Gout, Ivan
PATENT ASSIGNEE(S): Ludwig Institute for Cancer Research, Barbados
SOURCE: PCT Int. Appl., 146 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | | DATE |
|---|---|---|---|---|---|
| WO 9321328 | A1 | 19931028 | WO 1993-GB761 | | 19930413 |
| W: AU, CA, JP, US | | | | | |
| RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE | | | | | |
| AU 9339017 | A1 | 19931118 | AU 1993-39017 | | 19930413 |
| AU 664893 | B2 | 19951207 | | | |
| EP 590126 | A1 | 19940406 | EP 1993-908028 | | 19930413 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE | | | | | |
| JP 06510207 | T2 | 19941117 | JP 1993-518112 | | 19930413 |
| JP 07203963 | A2 | 19950808 | JP 1994-4313 | | 19940120 |
| JP 3471879 | B2 | 20031202 | | | |
| US 5824492 | A | 19981020 | US 1994-162081 | | 19940207 |
| US 5846824 | A | 19981208 | US 1997-780872 | | 19970109 |
| US 6274327 | B1 | 20010814 | US 1998-85957 | | 19980527 |
| PRIORITY APPLN. INFO.: | | | GB 1992-8135 | A | 19920413 |
| | | | WO 1993-GB761 | A | 19930413 |
| | | | US 1994-162081 | A3 | 19940207 |
| | | | US 1997-780872 | A3 | 19970109 |

AB PI3 kinase activity is manufactured by expression of the cloned genes in insect cell culture. Affinity purification of the protein from bovine brain using Y751 phosphopeptide from PDGF-β receptor as the affinity ligand identified the p85 and p110 (actual mol. weight 124 kDa) proteins that bound to the column with very high affinity and appeared to form a complex. A cDNA bank from SGBAF-1 cells in λUni-ZAP was screened with amino acid sequence-derived oligonucleotide probes from the p110 protein. and the cDNA expressed in Sf9 cells using a baculovirus vector. A cDNA clone was expressed in Sf9 cells using a baculovirus vector to give a detectable PI3 kinase activity. Tissue specificity of gene expression is determined and the corresponding human cDNA was cloned.

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**Search Results** - Record(s) 1 through 10 of 10 returned.

1. US 6046049A. New antisense compounds targeting nucleic acids encoding human PI3 kinase p110 delta useful for treating a disease or condition associated with PI3 kinase p110 delta expression, e.g. rheumatoid arthritis, asthma. COWSERT, L M, et al. A61K031/7125 A61K048/00 A61P011/06 A61P019/02 A61P029/00 A61P043/00 C07H021/04 C12N015/00 C12N015/09 C12N015/11 C12Q001/68.

2. 6046049. 19 Jul 99; 04 Apr 00. Antisense modulation of PI3 kinase p110 delta expression. Monia; Brett P., et al. 435/375; 435/366 435/6 435/91.1 536/23.1 536/24.31 536/24.33 536/24.5. C07H021/04 C12Q001/68 C12N015/00.

3. 5846824. 09 Jan 97; 08 Dec 98. Polypeptides having kinase activity, their preparation and use. Hiles; Ian D., et al. 435/348; 435/320.1 435/325 536/23.2 536/24.3. C12N005/10 C12N005/16 C12N015/54 C12N015/63.

4. 6274327. 27 May 98; 14 Aug 01. Polypeptides having kinase activity, their preparation and use. Hiles; Ian D., et al. 435/7.1; 435/15 435/194 435/21 435/252.3 435/348 435/6 435/69.1 435/69.2 435/7.2 435/7.8 530/350 530/388.26. G01N033/53 C12Q001/68 C12Q001/48 C12Q001/42.

5. 5824492. 07 Feb 94; 20 Oct 98. Polypeptides having kinase activity, their preparation and use. Hiles; Ian D., et al. 435/15; 435/194 435/29. C12N009/12 C12Q001/48.

6. 20010016332. 07 Aug 97. 23 Aug 01. AGE-1 POLYPEPTIDES AND RELATED MOLECULES AND METHODS. RUVKUN, GARY, et al. 435/69.1; 424/9.1 435/320.1 435/325 435/375 800/21 800/3 800/8 C12N015/63 A61K049/00 C12N005/00.

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